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Which psychological factors exacerbate Irritable Bowel Syndrome? Development of a comprehensive model

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Abstract

Objective—There is evidence that psychological factors affect the onset, severity and duration of Irritable Bowel Syndrome (IBS). However, it is not clear which psychological factors are the most important and how they interact. The aims of the current study are to identify the most important psychological factors predicting IBS symptom severity and to investigate how these psychological variables are related to each other.

Methods—Study participants were 286 IBS patients who completed a battery of psychological questionnaires including neuroticism, abuse history, life events, anxiety, somatization and catastrophizing. IBS severity measured by the IBS Severity Scale was the dependent variable. Path analysis was performed to determine the associations among the psychological variables, and IBS severity.

Results—Although the hypothesized model showed adequate fit, post hoc model modifications were performed to increase prediction. The final model was significant (Chi^2 = 2.2; p=0.82; RMSEA < .05) predicting 36% of variance in IBS severity. Catastrophizing (Standardized coefficient (β)=0.33; p <.001) and Somatization (β=0.20; p <.001) were the only two psychological variables directly associated with IBS severity. Anxiety had an indirect effect on IBS symptoms through catastrophizing (β=0.80; p <.001); as well as somatization (β=0.37; p <. 001). Anxiety, in turn, was predicted by neuroticism (β=0.66; p<.001) and stressful life events (β=0.31; p<.001).

Conclusion—While cause-and-effect cannot be determined from these cross-sectional data, the outcomes suggest that the most fruitful approach to curb negative effects of psychological factors on IBS is to reduce catastrophizing and somatization.

Keywords

Irritable Bowel Syndrome; Psychological distress; Somatization; Catastrophizing; Life Events; Neuroticism

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Introduction

Irritable Bowel Syndrome (IBS) is a common, chronic gastrointestinal disorder, characterized by recurring episodes of abdominal pain associated with altered bowel habits[1]. There is no consensus on the etiology of IBS but biological, psychological, and sociological factors are all believed to contribute to the onset, severity, and natural history of the disorder. Psychological factors appear to play particularly important roles as moderators of symptom severity, symptom persistence, decisions to seek treatment, and response to treatment[2-4], but it is not clear which psychological factors are the most important in explaining these outcomes. In a recent review 23 different psychosocial factors were identified that are associated with IBS[5], indicating the richness of this literature and the variability in factors identified. The psychological concepts most commonly associated with IBS are listed below:

Stress

The effect of stress on IBS is almost universally recognized by clinicians and patients. IBS symptoms wax and wane with daily stress[6, 7] and IBS patients report more lifetime stressful events than healthy controls[8]. There is particularly strong evidence for the role of early life stressors such as sexual abuse and maternal separation in IBS[9-12]. Dysfunctional brain-gut interactions have been found in maternally separated rodents-an often studied model of early life stress in IBS[13]. Moreover IBS patients show greater reactivity to stress[14]; that is, compared to healthy controls, the same exposure to stress leads to a greater physiological gut response in IBS patients.

Personality

Certain personality traits or temperament characteristics may make one more vulnerable to the effects of stressors. A widely studied personality factor is neuroticism[15], which describes people who readily experience negative affect. Subjects high in neuroticism are more reactive to stress and have stronger reactions to recurring problems. Neuroticism is one of the few personality traits that has been consistently found to be increased in IBS patients compared to controls[16-20].

Coping

The way patients cope with stress and pain mediates health outcomes[21]. One of the most robust predictors of pain intensity is a coping strategy named Pain Catastrophizing, defined as a maladaptive way of coping (or not coping) with pain by magnifying the threat or seriousness of pain and feeling helpless to do anything about it[22]. Catastrophizing is associated with more intense pain and greater disability in pain patients, including those who suffer from IBS[22-25].

Psychological distress refers to feeling anxious and depressed. These symptoms are more frequent and more intense in IBS patients, and they are associated with more gastrointestinal symptoms, disability and quality of life impairment[7, 26-31]. In 30% to 90%[32-34] of IBS patients, psychological symptoms are so severe that co-morbid psychiatric disorders can be diagnosed. The association between psychological distress and IBS seems to be bidirectional in nature: psychological distress both precedes the onset of IBS[35, 36], and is aggravated by the challenges of managing a chronic gastrointestinal disorder[37].

Somatization refers to the psychological tendency to report multiple physical symptoms. Somatization is frequently seen in IBS patients[32], many of whom receive diagnoses of other functional gastrointestinal disorders, chronic pain syndromes, and symptoms such as chronic fatigue, frequent urination, bad breath and heart palpitations. The overlap between IBS and

these other co-morbid disorders and symptoms does not appear to be explained by a common pathophysiology[34]. Instead people high in somatization are thought to be hypervigilant to noticing somatic sensations and to attach disease significance to these symptoms[32, 34].

It has been established that these psychological variables play a role in IBS, but it is essential to determine the relative strength of their contribution to the waxing and waning of IBS as this will suggest which psychological factors should be targeted in treatment. Besides the direct effects these psychological factors have on IBS outcomes, they are also associated with each other. For example, the effect of neuroticism on IBS seems to be mediated by anxiety[19] and in functional dyspepsia it has been shown that psychological distress is associated with somatization[38]. Thus, we devised a model (see Figure 1) in which these associations between psychological factors were taken into account. Specifically, we hypothesize that stressful life events, abuse history and neuroticism aggravate maladaptive coping, anxiety, and somatization, which in turn influence IBS symptom severity. The aims of the current study were to test our model, to identify the most important psychological factors predicting IBS symptom severity, and to investigate how these psychological variables are related to each other.

Methods

Study Design

This study analyzes data from a study on the pathophysiology of IBS[39, 40]. For this study subjects were admitted to a research clinic at the University of North Carolina for a 24- to 30-h period. On the day of admission the questionnaires described below were completed. Data from all participants were used in the current analyses.

Subjects

Subjects were recruited by advertisements or physician referrals and screened by telephone. The study was approved by the institutional review board of the University of North Carolina (UNC) and all subjects provided informed consent. The study population consisted of patients with a physician diagnosis of IBS who also met Rome II or III criteria for IBS (depending on time of enrollment, 77% of patients were screened with Rome II criteria) and had current symptom activity (abdominal pain at least once a week in the past month). These subjects had no history of gastrointestinal resection (other than appendectomy or cholecystectomy), known Inflammatory Bowel Disease, coeliac disease, lactose malabsorption, heart disease, or diabetes mellitus, and they were not pregnant at the time of study.

Measures

IBS severity—The Irritable Bowel Syndrome Severity Scale (IBS-SS) is a validated questionnaire containing five questions which address severity and frequency of abdominal pain, bloating severity, dissatisfaction with bowel habits, and IBS impact on everyday activities[41]. All questions are scored on a scale ranging from 0–100 and contribute equally to the total score. Previous studies have established that scores below 175 represent mild IBS symptoms, 175–300 represents moderate severity, and scores above 300 represent severe IBS[41].

Sexual and physical abuse—Abuse history was measured by the Sexual and Physical Abuse History Questionnaire developed for use in functional gastrointestinal disorders by Leserman and Drossman[42]. Patients are asked to indicate if they have ever been sexually abused (had someone touch their sex organs against their will, were made to touch someone

else's sex organs, were forced to have sex) or physically abused (having been hit/kicked/beaten or someone seriously threatened their life). A subject is assumed to have a history of abuse if they reported 'yes' to at least one of these items over their life time.

Life events—The Family Inventory of Life Events scale (FILE)[43] is a measure of individual stressful life events as well as life events within the immediate family (e.g., spouse, children). The FILE records 67 life events within a person's nuclear family over the past year. Events are included that require a significant change or adjustment within the family such as 'mother giving birth', 'family member assaulted or robbed', or 'family member quit work due to health reason'. A total score of cumulative stressful life events was obtained by counting the number of reported events.

Neuroticism—The NEO personality inventory[15] consists of 5 subscales, but only the Neuroticism subscale was used in this study. Forty-eight items are included in this subscale which ask about negative emotions (e.g., susceptibility to experience anger, guilt or anxiety) and stress reactivity. All items are answered on a 5 point scale and summed to obtain a total score.

Anxiety—Anxiety was measured by the anxiety subscale of the *Brief Symptom Inventory-18* (BSI-18)[44]. The subscale consists of 6 items asking about anxious cognitions (e.g., "feeling fearful") over the past 7 days. These items are rated on a 5-point scale ranging from "not at all" to "extremely".

Somatization—Somatization was determined with the *Brief Symptom Inventory-18* (BSI-18)[44]. The 6-item somatization subscale asks the subject to rate how often they were bothered by somatic symptoms such as "Feeling weak in parts of your body" or "Trouble getting your breath". The somatization subscale of the BSI contains one gastrointestinal symptom item, nausea or upset stomach; we excluded this item from the scale.

Catastrophizing—The catastrophizing scale (6 items) of the *Coping Strategies Scale*[45] was used. This scale reflects feelings of hopelessness and the expectation that pain will become worse. It shows good reliability and validity in a sample of pain patients[45] and has been used in studies on IBS[24, 25].

Data analysis

Path analysis was implemented with IBM® SPSS® AMOS 19.9, which was used to test the model shown in Figure 1. Path analysis allows for testing all associations between factors at the same time. Chi-square was used for an overall test of the model ($p > .05$ denotes significance). A significant model can be improved by removing non-significant pathways. We trimmed non-significant paths one by one, each time testing if cutting would create a non-significant overall model. If the overall model became non-significant the non-significant pathway would not be removed.

We fit separate models using the total IBS-SS scores as well as individual IBS-SS scores on pain frequency and disability but all three dependent variables generated largely the same significant paths, so the model for overall IBS-SS score is depicted here. Goodness of fit was determined by the Root Mean Square Error of Approximation (RMSEA) with <.05 denoting close fit.

To test the robustness of our trimmed model we tested if alternative models yielded a better goodness of fit index. Alternative models were created by substituting reciprocal direct effects for other types of paths. For example, if the final model specified Anxiety →

Somatization, alternative models were tested in which this association was substituted by Somatization → Anxiety or Anxiety ↔ Somatization. With 7 variables in the model we have sufficient sample size to test the model (N should be at least 10 to 20 times the number of variables in the model)[46].

Results

Sample characteristics

A total of 286 IBS patients participated. Mean age was 34.6 years (sd=11.7; age range 18–73). Participants were predominantly female (81.5%), and race was Caucasian (71.3%), African American (19.2%), or Hispanic (3.5%). IBS severity was predominantly moderate (43%) to high (39.5%). Means and standard deviations of all variables in our model are given in Table 1.

Path Model

The assumptions for path analysis were evaluated. One outlier (defined as z-scores scores in excess of 3.29[47]) was found for the FILE score and one for the Catastrophizing score; these two subjects were deleted from the analysis. Normality was not violated (except for abuse which was a dichotomous variable). Missing values were replaced by maximum likelihood estimates.

The hypothesized model showed adequate fit to the data: Chi^2 (df=1)= 0.29; p=0.56; RMSEA < .05. Post hoc model modifications whereby non-significant associations were removed, were performed to develop a trimmed model. The final model is given in Figure 2 (Chi^2 (df=5)= 2.2; p=0.82; RMSEA < .05. Several alternative models –which included small model modifications- were tested and all these models became nonsignificant.

As can be seen in Figure 2, catastrophizing and somatization were the only two psychological variables directly associated with IBS severity. The significant positive coefficients indicate that increases in catastrophizing and somatization were associated with increases in IBS symptom severity. Additionally, anxiety had an indirect effect on IBS symptoms through catastrophizing as well as somatization -- while anxiety was in turn predicted by neuroticism and life events. Thus, the model shows that neuroticism and stressful life events increase anxiety. The effect of anxiety on IBS severity is mediated by catastrophizing and somatization.

Additional associations in the model were: (a) A positive effect of catastrophizing and life events on somatization; and (b) a negative effect of catastrophizing on anxiety. Combined these variables predicted 36% of the variance in IBS severity.

Discussion

The aim of the current study was to develop a model of psychological influences on IBS symptoms that can guide research and therapy. We observed that the two most important variables associated with IBS severity are the maladaptive coping style referred to as catastrophizing and somatization. The effects of all other psychological variables seem to be explained by these two factors. However, anxiety had an important intermediate place in the model because it was associated with both increases in catastrophizing and somatization. The role of other psychological variables such as neuroticism and life stressors was limited to being associated with increases in anxiety. Although the model may suggest cause-effect associations, we acknowledge that the current study is correlational so we cannot claim that any of the psychological factors are causally related to each other or to IBS outcomes. Prospective longitudinal studies are needed to confirm the model.

The model suggests that the most fruitful approach to curb the effects of psychological factors on IBS is to reduce catastrophizing and somatization. For catastrophizing, the common adage to 'reduce stress' is applicable. But rather than reducing stress by diminishing exposure to stressors (such as certain job and family responsibilities, which may be hard to avoid) the aim should be to improve coping with stress. There are existing treatment techniques, largely used within Cognitive Behavioral Therapy (CBT), that are focused on replacing maladaptive coping strategies, specifically catastrophizing, with more positive cognitions and behaviors. In fact, CBT has been found to be an effective treatment for IBS, not only reducing IBS symptoms but also reducing somatization and catastrophizing as well as improving mood[48-50]. As expected from our model, most studies have found that treatment outcomes with CBT are not mediated by improvements in mood[48, 50-52] although one study did find evidence for this[53]. Rather, two treatment studies have confirmed the important role of coping in reducing IBS symptoms[54, 55]. This supports our model, giving coping a more important role in predicting IBS outcomes than anxiety.

Besides a focus on improving coping, our model suggests that reducing somatization should be effective in ameliorating IBS symptoms. In our model, somatization was included as a predictor of IBS severity, but it may also be considered an outcome by itself. IBS patients can suffer from several bodily symptoms including gastrointestinal and non-gastrointestinal symptoms. Not surprisingly the same psychological factors in our model that predict IBS severity also predict somatization. These include anxiety, stressful life events, and catastrophizing. By reducing these factors, we may not only reduce IBS symptoms but also other non-gastrointestinal symptoms. Both somatization and IBS symptom severity have been found to be responsive to treatment with hypnosis, although the mechanisms by which this occurs is not clear[56-58]. This suggests that hypnosis may be a particularly helpful treatment in reducing multiple symptoms. Thus, psychological treatments are available – CBT and hypnosis – which target the two psychological variables with the greatest impact on IBS symptom severity.

Of all the psychological variables in the original model (see Figure 1), only lifetime sexual/physical abuse was not related to the other psychological variables nor to IBS severity. This is a surprising finding given the large literature on the association between abuse and IBS[11]. Most of these previous studies, however, have shown that rates of abuse are increased in IBS patients compared to healthy controls. This does not have direct relevance to our current findings in which we investigated IBS severity rather than differences between IBS and controls. Very few have studied the influence of abuse history on IBS severity. One study found that patients with a history of sexual abuse reported higher pain in general-but did not distinguish abdominal pain from other types of pain[59]. Two studies found no increase in abdominal pain, constipation or diarrhea[60, 61], although a small decrease was found in bloating among those who have been sexually abused. Thus, there is little evidence from the existing literature that abuse history affects IBS symptom severity which is in line with our current findings. However, there is a large literature suggesting that abuse history is associated with increased anxiety[62, 63], so our lack of association between these variables suggests that more research is needed before excluding the role of abuse on distress and symptoms in IBS. For example, abuse severity or cumulative exposure to abuse may be more important than abuse history in predicting IBS severity.

There is one other pathway in our model that does not make intuitive sense: The reciprocal association between catastrophizing and anxiety. It can be expected that these variables are positively associated: An anxious person would be more likely to catastrophize in the face of pain; while worrying that the pain is going to become worse and there is nothing you can do, will likely increase anxiety. Indeed we found that anxiety increases catastrophizing, but catastrophizing, in turn, was associated with decreases in anxiety. It may not be immediately

clear how maladaptive coping reduces anxiety until we place catastrophizing in a larger context. As proposed by Sullivan et al.[22] and empirically validated among IBS patients by Lackner et al.[64], catastrophizing may not have the primary intent to reduce pain but rather to increase social support. In order to solicit social support, patients may report more pain and display more pain behaviors[64]. It may be argued that soliciting social support for pain may be an effective strategy to reduce pain. If the pain becomes too intense to handle or exceeds coping abilities, patients may look to others for help; a coping strategy common among children with pain who solicit their parents for help and whose pain outcomes are dependent on parental coping strategies[65]. Thus, it can be hypothesized that when IBS patients feel overwhelmed by pain they may display catastrophizing behaviors –temporarily increasing the pain- in order to elicit help and support by others. By delegating responsibility to cope with pain, distress may go down. More studies are needed to determine if catastrophizing is used to increase social support for pain or if another explanation is of more relevance.

We would like to compare our results with other studies, but surprisingly there are few studies testing the relative influence of psychological variables on IBS. There have been some studies employing structural equation modeling or path analysis to investigate the role of several physiological and psychological factors in IBS. Naliboff and colleagues[66] investigated whether health related quality of life was determined by gastrointestinal symptoms or psychological symptoms. Only one global psychological construct was investigated: overall psychological distress as measured by the Symptom Checklist 90[67]. The authors concluded that psychological distress had a stronger impact on quality of life than gastrointestinal symptoms in patients with IBS. Lackner and colleagues[51] investigated the pathways through which cognitive behavioral therapy (CBT) affects IBS improvement. They concluded that CBT has a direct effect on gastrointestinal symptom improvement but no significant effect on psychological distress. Of interest was their finding that psychological distress had a greater impact on IBS quality of life than did gastrointestinal symptom improvement. Garland and colleagues[55] tested therapeutic mediators of the effect of Mindfulness Training on IBS outcomes. They found that both pain sensitivity and coping (catastrophizing and reinterpretation of pain sensations) had independent effects on IBS severity and quality of life. Thus, these studies establish the importance of psychological distress and coping for IBS outcomes, but none of the models included both factors. Our current findings support and extend these observations by showing that the effects of psychological distress (as measured by anxiety) on IBS outcomes are mediated by coping.

Our study has several limitations. The psychological factors included in our model were chosen based on a literature review at the beginning of our study. We chose variables for which there was a large evidence base and did not show too much overlap with each other. This selection process can create bias. Despite our best efforts to include the most relevant psychological variables we may have left out some important and/or newer concepts such as perceived stress[68, 69], gastro-intestinal specific anxiety[70], anxiety sensitivity[19], alexithymia[68, 71], or hypervigilance[72, 73] to symptoms. Models that include these variables may show a different outcome. Therefore, studies are needed to replicate and extend our current findings. Furthermore, as mentioned before, the current retrospective dataset does not allow for cause-effect inferences between factors. Longitudinal studies are needed to test whether change in one factor is followed by change in another. In addition, our data is limited by the fact that we included a self-selected sample who all had consulted a physician for their symptoms. The decision to see a doctor may be driven by psychological factors rather than severity of the gastrointestinal symptoms[74, 75]. This suggests that psychological factors may play a more important role in our sample. Alternatively, symptoms may interfere less with life among non-consulting patients due to reduced levels of stress, anxiety, catastrophizing etc. in this population. Therefore, our model is in need of replication in non-consulting samples before generalization to the whole population can be assumed.

Moreover we acknowledge that our model is limited by its exclusive focus on psychological factors. We realize that models need to be tested that also include physiological variables such as motility and social factors such as gender and social support, but to keep the model simple these interactions have been omitted from the current analysis. We believe these limitations to the model are justified by previously cited evidence that psychological variables are the strongest moderators of symptom severity[51, 55, 66]. The findings of our study may help devise future studies which include all biopsychosocial aspects, by providing a better understanding of the most important psychological factors to be included. A final limitation to our study is that it focuses only on current symptom severity and ignores other outcomes such as symptom persistence and responsiveness to treatment. The cross-sectional nature of the research design does not allow us to examine these delayed consequences but clearly more data is needed to determine the relative impact of psychological factors on these outcomes.

In conclusion, the present study is the first to test an overarching model explaining the role of psychological factors in IBS. It includes both past life experiences, personality and more modifiable psychological factors. Of all these factors, catastrophizing and somatization seem to be the most important in regard to IBS symptom severity and deserve particular attention in research and treatment. It needs to be acknowledged that physiological and social factors are also of importance in predicting IBS severity and interact with psychological factors to predict IBS outcomes. More studies are needed in which all variables are tested simultaneously. We hope that the current model can be a guideline for future research and treatment of IBS.

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References

1. Drossman, DA.; Corazziari, E.; Delvaux, M., et al. Rome III: The Functional Gastrointestinal Disorders. McLean, Virginia: Degnon Associates; 2006.
2. Drossman DA, Thompson WG. The Irritable-Bowel-Syndrome - Review and A Graduated Multicomponent Treatment Approach. Annals of Internal Medicine. 1992; 116:1009–1016. [PubMed: 1586090]
3. Drossman DA. Do psychosocial factors define symptom severity and patient status in irritable bowel syndrome? Am J Med. 1999; 107:41S–50S. [PubMed: 10588172]
4. Drossman DA, Chang L, Bellamy N, et al. Severity in irritable bowel syndrome: a Rome Foundation Working Team report. Am J Gastroenterol. 2011; 106:1749–1759. quiz 1760. [PubMed: 21747417]
5. Surdea-Blaga T, Baban A, Dumitrascu DL. Psychosocial determinants of irritable bowel syndrome. World J Gastroenterol. 2012; 18:616–626. [PubMed: 22363132]
6. Blanchard EB, Lackner JM, Jaccard J, et al. The role of stress in symptom exacerbation among IBS patients. J Psychosom Res. 2008; 64:119–128. [PubMed: 18222125]
7. Levy RL, Cain KC, Jarrett M, et al. The relationship between daily life stress and gastrointestinal symptoms in women with irritable bowel syndrome. Journal of Behavioral Medicine. 1997; 20:177–193. [PubMed: 9144039]
8. Whitehead WE, Crowell MD, Robinson JC, et al. Effects of Stressful Life Events on Bowel Symptoms - Subjects with Irritable-Bowel-Syndrome Compared with Subjects Without Bowel Dysfunction. GUT. 1992; 33:825–830. [PubMed: 1624167]
9. Klooker TK, Braak B, Painter RC, et al. Exposure to severe wartime conditions in early life is associated with an increased risk of irritable bowel syndrome: a population-based cohort study. Am J Gastroenterol. 2009; 104:2250–2256. [PubMed: 19513027]

10. Chitkara DK, van Tilburg MA, Blois-Martin N, et al. Early life risk factors that contribute to irritable bowel syndrome in adults: a systematic review. Am.J Gastroenterol. 2008; 103:765–774. [PubMed: 18177446]
11. Leserman J, Drossman DA. Relationship of abuse history to functional gastrointestinal disorders and symptoms: some possible mediating mechanisms. Trauma Violence Abuse. 2007; 8:331–343. [PubMed: 17596349]
12. van Tilburg MA, Runyan DK, Zolotor AJ, et al. Unexplained gastrointestinal symptoms after abuse in a prospective study of children at risk for abuse and neglect. Ann Fam Med. 2010; 8:134–140. [PubMed: 20212300]
13. O'Mahony SM, Hyland NP, Dinan TG, et al. Maternal separation as a model of brain-gut axis dysfunction. Psychopharmacology (Berl). 2011; 214:71–88. [PubMed: 20886335]
14. Mayer EA, Naliboff BD, Chang L, et al. V. Stress and irritable bowel syndrome. Am J Physiol Gastrointest Liver Physiol. 2001; 280:G519–G524. [PubMed: 11254476]
15. Costa, PT., Jr.; McCrae, RR. NEO PI-R professional manual. Odessa, FL: Psychological Assessment Resources, Inc; 1992.
16. Farnam A, Somi MH, Sarami F, et al. Personality factors and profiles in variants of irritable bowel syndrome. World J Gastroenterol. 2007; 13:6414–6418. [PubMed: 18081232]
17. Tosic-Golubovic S, Miljkovic S, Nagorni A, et al. Irritable bowel syndrome, anxiety, depression and personality characteristics. Psychiatr Danub. 2010; 22:418–424. [PubMed: 20856185]
18. Tkalcic M, Hauser G, Stimac D. Differences in the health-related quality of life, affective status, and personality between irritable bowel syndrome and inflammatory bowel disease patients. Eur J Gastroenterol Hepatol. 2010; 22:862–867. [PubMed: 19701092]
19. Hazlett-Stevens H, Craske MG, Mayer EA, et al. Prevalence of irritable bowel syndrome among university students: the roles of worry, neuroticism, anxiety sensitivity and visceral anxiety. J.Psychosom.Res. 2003; 55:501–505. [PubMed: 14642979]
20. Talley NJ, Boyce PM, Jones M. Is the association between irritable bowel syndrome and abuse explained by neuroticism? A population based study. GUT. 1998; 42:47–53. [PubMed: 9505885]
21. Lazarus, RS.; Folkman, S. Stress, appraisal, and coping. New York: Springer; 1984.
22. Sullivan MJ, Thorn B, Haythornthwaite JA, et al. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain. 2001; 17:52–64. [PubMed: 11289089]
23. Lackner JM, Quigley BM, Blanchard EB. Depression and abdominal pain in IBS patients: the mediating role of catastrophizing. Psychosom Med. 2004; 66:435–441. [PubMed: 15184708]
24. Lackner JM, Quigley BM. Pain catastrophizing mediates the relationship between worry and pain suffering in patients with irritable bowel syndrome. Behav Res Ther. 2005; 43:943–957. [PubMed: 15896288]
25. Lackner J, Jaccard J, Baum C, et al. Patient-reported outcomes for irritable bowel syndrome are associated with patients' severity ratings of gastrointestinal symptoms and psychological factors. Clin Gastroenterol Hepatol. 2011; 9:957–964. e1. [PubMed: 21699821]
26. Koloski NA, Boyce PM, Jones MP, et al. What level of IBS symptoms drives impairment in health-related quality of life in community subjects with irritable bowel syndrome? Are current IBS symptom thresholds clinically meaningful? Qual Life Res. 2012; 21:829–836. [PubMed: 21833813]
27. Koloski NA, Talley NJ, Boyce PM. Does psychological distress modulate functional gastrointestinal symptoms and health care seeking? A prospective, community Cohort study. Am J Gastroenterol. 2003; 98:789–797. [PubMed: 12738457]
28. Lackner JM, Jaccard J, Blanchard EB. Testing the sequential model of pain processing in irritable bowel syndrome: a structural equation modeling analysis. Eur J Pain. 2005; 9:207–218. [PubMed: 15737813]
29. Okami Y, Kato T, Nin G, et al. Lifestyle and psychological factors related to irritable bowel syndrome in nursing and medical school students. J Gastroenterol. 2011; 46:1403–1410. [PubMed: 21863219]
30. Seres G, Kovacs Z, Kovacs A, et al. Different associations of health related quality of life with pain, psychological distress and coping strategies in patients with irritable bowel syndrome and

inflammatory bowel disorder. J Clin Psychol Med Settings. 2008; 15:287–295. [PubMed: 19104985]

31. Choung RS, Locke GR 3rd, Zinsmeister AR, et al. Psychosocial distress and somatic symptoms in community subjects with irritable bowel syndrome: a psychological component is the rule. Am J Gastroenterol. 2009; 104:1772–1779. [PubMed: 19491833]
32. Whitehead WE, Palsson O, Jones KR. Systematic review of the comorbidity of irritable bowel syndrome with other disorders: What are the causes and implications? Gastroenterology. 2002; 122:1140–1156. [PubMed: 11910364]
33. Drossman DA, Camilleri M, Mayer EA, et al. AGA technical review on irritable bowel syndrome. Gastroenterology. 2002; 123:2108–2131. [PubMed: 12454866]
34. Whitehead WE, Palsson OS, Levy RR, et al. Comorbidity in irritable bowel syndrome. Am.J.Gastroenterol. 2007; 102:2767–2776. [PubMed: 17900326]
35. Nicholl BI, Halder SL, Macfarlane GJ, et al. Psychosocial risk markers for new onset irritable bowel syndrome--results of a large prospective population-based study. Pain. 2008; 137:147–155. [PubMed: 17928145]
36. Gwee KA, Graham JC, McKendrick MW, et al. Psychometric scores and persistence of irritable bowel after infectious diarrhoea. Lancet. 1996; 347:150–153. [PubMed: 8544549]
37. Smith RC, Greenbaum DS, Vancouver JB, et al. Psychosocial factors are associated with health care seeking rather than diagnosis in irritable bowel syndrome. Gastroenterology. 1990; 98:293–301. [PubMed: 2295384]
38. Jones MP, Coppens E, Vos R, et al. A multidimensional model of psychobiological interactions in functional dyspepsia: a structural equation modelling approach. GUT. 2012
39. Dorn SD, Palsson OS, Thiwan SI, et al. Increased colonic pain sensitivity in irritable bowel syndrome is the result of an increased tendency to report pain rather than increased neurosensory sensitivity. GUT. 2007; 56:1202–1209. [PubMed: 17483191]
40. Kanazawa M, Palsson OS, Thiwan SI, et al. Contributions of Pain Sensitivity and Colonic Motility to IBS Symptom Severity and Predominant Bowel Habits. Am J Gastroenterol. 2008; 103:2550–2561. [PubMed: 18684175]
41. Francis CY, Morris J, Whorwell PJ. The irritable bowel severity scoring system: a simple method of monitoring irritable bowel syndrome and its progress. Alimentary Pharmacology & Therapeutics. 1997; 11:395–402. [PubMed: 9146781]
42. Leserman J, Drossman DA, Li Z. The reliability and validity of a sexual and physical abuse history questionnaire in female patients with gastrointestinal disorders. Behav.Med. 1995; 21:141–150. [PubMed: 8789650]
43. McCubbin, H.; Patterson, J.; Wilson, L. Family Inventory of Life Events and Changes (FILE). In: McCubbin, HI.; Thompson, AI.; McCubbin, MA., editors. Family Assessment: Resiliency, coping and adaptation-inventories for research and practice. Madison: University of Wisconsin System; 1996. p. 103-178.
44. Derogatis, LR. BSI 18 Brief Symptom Inventory 18: Administration, scoring, and procedures manual. NCS Pearson,Inc; 2000.
45. Keefe FJ, Brown GK, Wallston KA, et al. Coping with rheumatoid arthritis pain: catastrophizing as a maladaptive strategy. Pain. 1989; 37:51–56. [PubMed: 2726278]
46. Kline, RB. Principles and Practice of Structural Equation Modeling. New York: Guilford Press; 2005.
47. Tabachnick, BG.; Fidell, LS. Using multivariate statistics. Fifth Edition. Boston MA: Pearson Education; 2007.
48. Lackner JM, Mesmer C, Morley S, et al. Psychological treatments for irritable bowel syndrome: a systematic review and meta-analysis. J Consult Clin Psychol. 2004; 72:1100–1113. [PubMed: 15612856]
49. Toner BB, Segal ZV, Emmott S, et al. Cognitive-behavioral group therapy for patients with irritable bowel syndrome. Int.J Group Psychother. 1998; 48:215–243. [PubMed: 9563239]
50. Kroenke K, Swindle R. Cognitive-behavioral therapy for somatization and symptom syndromes: a critical review of controlled clinical trials. Psychother Psychosom. 2000; 69:205–215. [PubMed: 10867588]

51. Lackner JM, Jaccard J, Krasner SS, et al. How does cognitive behavior therapy for irritable bowel syndrome work? A mediational analysis of a randomized clinical trial. Gastroenterology. 2007; 133:433–444. [PubMed: 17681164]
52. Reme SE, Stahl D, Kennedy T, et al. Mediators of change in cognitive behaviour therapy and mebeverine for irritable bowel syndrome. Psychol Med. 2011:1–11. [PubMed: 21477419]
53. Jones M, Koloski N, Boyce P, et al. Pathways connecting cognitive behavioral therapy and change in bowel symptoms of IBS. J Psychosom Res. 2011; 70:278–285. [PubMed: 21334499]
54. Oerlemans S, van Cranenburgh O, Herremans PJ, et al. Intervening on cognitions and behavior in irritable bowel syndrome: A feasibility trial using PDAs. J Psychosom Res. 2011; 70:267–277. [PubMed: 21334498]
55. Garland EL, Gaylord SA, Palsson O, et al. Therapeutic mechanisms of a mindfulness-based treatment for IBS: effects on visceral sensitivity, catastrophizing, and affective processing of pain sensations. J Behav Med. 2011
56. Palsson OS, Turner MJ, Johnson DA, et al. Hypnosis treatment for severe irritable bowel syndrome: investigation of mechanism and effects on symptoms. Dig.Dis.Sci. 2002; 47:2605–2614. [PubMed: 12452403]
57. Tan G, Hammond DC, Joseph G. Hypnosis and irritable bowel syndrome: a review of efficacy and mechanism of action. Am.J.Clin.Hypn. 2005; 47:161–178. [PubMed: 15754863]
58. Calvert EL, Houghton LA, Cooper P, et al. Long-term improvement in functional dyspepsia using hypnotherapy. Gastroenterology. 2002; 123:1778–1785. [PubMed: 12454833]
59. Leserman J, Drossman DA, Li ZM, et al. Sexual and physical abuse history in gastroenterology practice: How types of abuse impact health status. Psychosomatic Medicine. 1996; 58:4–15. [PubMed: 8677288]
60. Heitkemper MM, Cain KC, Burr RL, et al. Is childhood abuse or neglect associated with symptom reports and physiological measures in women with irritable bowel syndrome? Biol Res Nurs. 2011; 13:399–408. [PubMed: 21196423]
61. Heitkemper M, Jarrett M, Taylor P, et al. Effect of sexual and physical abuse on symptom experiences in women with irritable bowel syndrome. Nurs Res. 2001; 50:15–23. [PubMed: 19785241]
62. Polusny MA, Follette VM. Long-Term Correlates of Child Sexual Abuse - Theory and Review of the Empirical Literature. Applied & Preventive Psychology. 1995; 4:143–166.
63. Cougle JR, Timpano KR, Sachs-Ericsson N, et al. Examining the unique relationships between anxiety disorders and childhood physical and sexual abuse in the National Comorbidity Survey-Replication. Psychiatry Res. 2010; 177:150–155. [PubMed: 20381878]
64. Lackner JM, Gurtman MB. Pain catastrophizing and interpersonal problems: a circumplex analysis of the communal coping model. Pain. 2004; 110:597–604. [PubMed: 15288400]
65. Lipani TA, Walker LS. Children's appraisal and coping with pain: relation to maternal ratings of worry and restriction in family activities. J.Pediatr.Psychol. 2006; 31:667–673. [PubMed: 15905417]
66. Naliboff BD, Kim SE, Bolus R, et al. Gastrointestinal and psychological mediators of health-related quality of life in IBS and IBD: a structural equation modeling analysis. Am J Gastroenterol. 2012; 107:451–459. [PubMed: 22085819]
67. Derogatis, LR. SCL-90-R: Administration, scoring, and procedures manual. Minneapolis MN: National Computer Systems; 1994.
68. Endo Y, Shoji T, Fukudo S, et al. The features of adolescent irritable bowel syndrome in Japan. J Gastroenterol Hepatol. 2011; 26(Suppl 3):106–109. [PubMed: 21443721]
69. Spence MJ, Moss-Morris R. The cognitive behavioural model of irritable bowel syndrome: a prospective investigation of patients with gastroenteritis. GUT. 2007; 56:1066–1071. [PubMed: 17324974]
70. Labus JS, Mayer EA, Chang L, et al. The central role of gastrointestinal-specific anxiety in irritable bowel syndrome: further validation of the visceral sensitivity index. Psychosom.Med. 2007; 69:89–98. [PubMed: 17244851]
71. Costin S, Petrar S, Dumitrascu DL. Alexithymia in romanian females with irritable bowel syndrome (IBS). J Psychosom Res. 2006; 61:426–426.

72. Posserud I, Svedlund J, Wallin J, et al. Hypervigilance in irritable bowel syndrome compared with organic gastrointestinal disease. J Psychosom Res. 2009; 66:399–405. [PubMed: 19379956]

73. Chapman S, Martin M. Attention to pain words in irritable bowel syndrome: increased orienting and speeded engagement. Br J Health Psychol. 2011; 16:47–60. [PubMed: 21226783]

74. Whitehead WE, Bosmajian L, Zonderman AB, et al. Symptoms of psychologic distress associated with irritable bowel syndrome. Comparison of community and medical clinic samples. Gastroenterology. 1988; 95:709–714. [PubMed: 3396818]

75. Drossman DA, McKee DC, Sandler RS, et al. Psychosocial factors in the irritable bowel syndrome. A multivariate study of patients and nonpatients with irritable bowel syndrome. Gastroenterology. 1988; 95:701–708. [PubMed: 3396817]

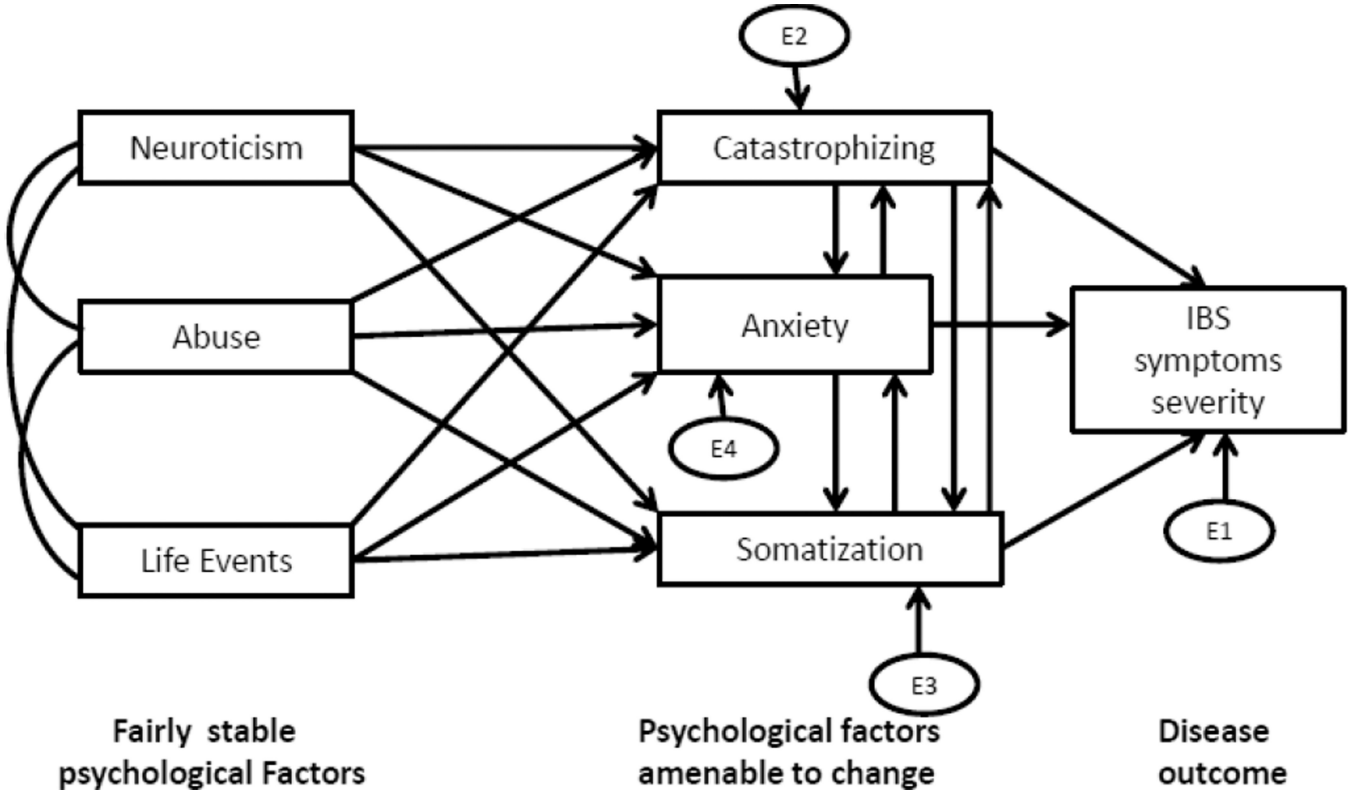

Figure 1.
Proposed model of psychological effects on IBS severity
Note: Direct effects between 'fairly stable psychological factors' and IBS severity were tested but for clarity not depicted in the current model

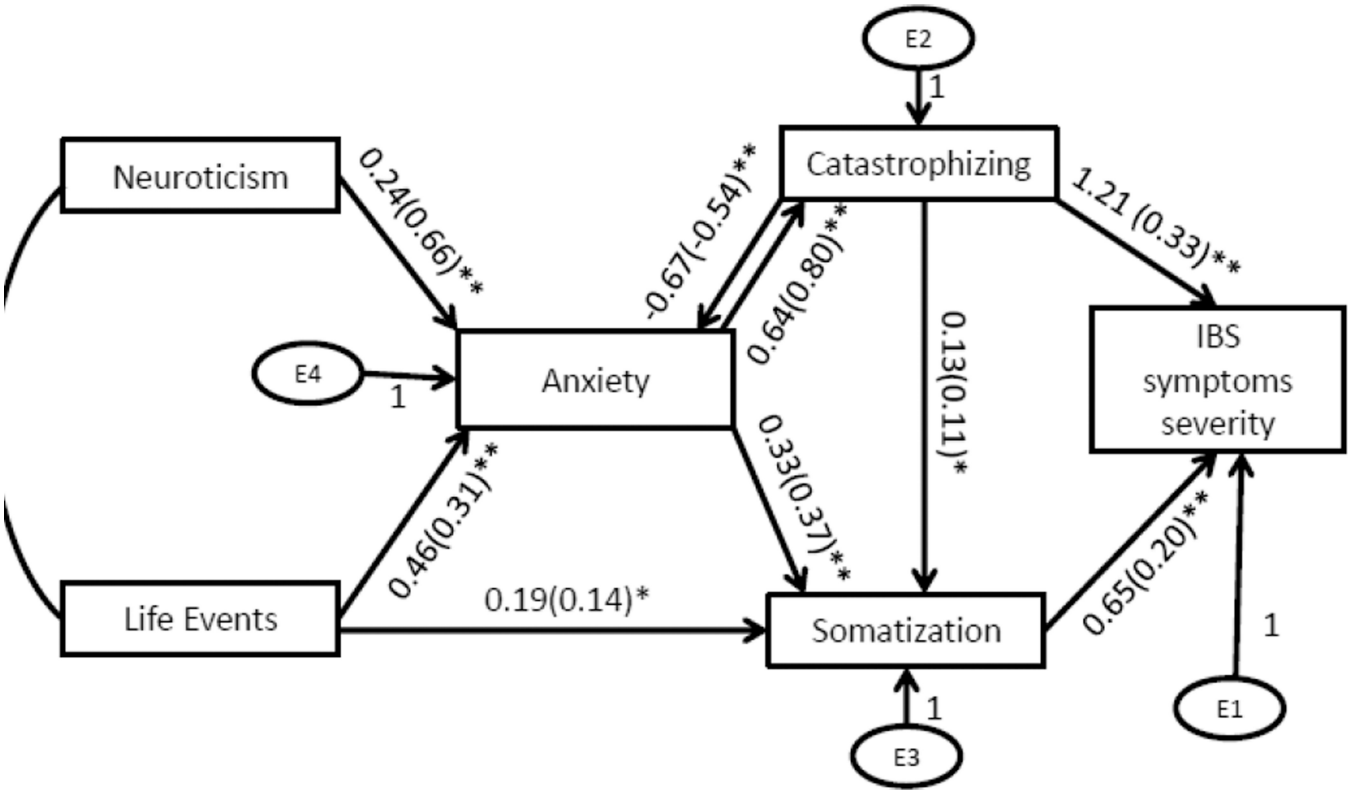

Figure 2.
Final model of psychological effects on IBS severity
Note: Numbers reflect unstandardized (standardized)coefficients. * P < .01, ** P < .001.

Table 1

Variables in the model

	M (SD) or %	Minimum and maximum score in the sample
Irritable Bowel Severity	270.0 (90.6)	50–500
Anxiety	51.7 (9.9)	38–81
Somatization	50.8 (9.0)	41–79
Catastrophizing	8.2 (7.9)	0–35
Neuroticism	63.6 (26.6)	1–99
Life events	9.3 (6.6)	0–31.4
History of abuse	54.3%	n/a